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DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 14:48:27 ON 02 SEP 1999
SEA (ALLOGENEIC OR SYNGENEIC) AND (TUMOR OR CANCER) AND
VACCIN?

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L1
VACCIN? QUE (ALLOGENEIC OR SYNGENEIC) AND (TUMOR OR CANCER) AND

FILE 'USPATFULL, CANCERLIT, EMBASE, MEDLINE, PROMT, SCISEARCH, BIOSIS,
CAPLUS, DRUGU, LIFESCI, TOXLINE, ADISALERTS, ADISINSIGHT, TOXLIT,
BIOTECHDS, AIDSLINE, JICST-EPLUS, DRUGNL, BIOBUSINESS, DRUGB, IFIPAT,
PHIN' ENTERED AT 14:50:46 ON 02 SEP 1999

L2 1080 S (ALLOGENEIC OR SYNGENEIC) AND (TUMOR OR CANCER) AND VACCIN?
A
L3 282 S L2 AND VACCIN?(10W)ADJUVANT?
L4 175 DUP REM L3 (107 DUPLICATES REMOVED)
L5 137 S L3 AND CELL(25W)VACCIN?
L6 94 DUP REM L5 (43 DUPLICATES REMOVED)
L7 1 S L3 AND BERD, D?/AU
E BERD, D?/AU
E BERD, DAVID/AU

Art Unit: 1642

melanoma tumor cell membranes with BCG which were used to treat skin clinic patient's at the second University of Vienna (page 658, col 2, para 4, the relevant sentence is underlined and is translated as "At the second Univeristy of Vienna's skin clinic, patients in Stage IIb were treated with autologous tumor cell membrane preparations with BCG. The reference teaches as disclosed above but does not teach the autologous tumor cell membrane preparation derived from DNP conjugated melanoma cells.

Definitely ~~103~~ 203 whatever

L4 ANSWER 22 OF 24 ADISALERTS COPYRIGHT 1999 (ADIS)

AN 1989:8381 ADISALERTS

DN 800592148

TI Clinical responses with active specific intralymphatic immunotherapy for **cancer** - a phase I-II trial

ADIS TITLE: **Cancer**: treatment.; Autologous **tumour** cell **vaccines**

AU Wiseman Cl; Rao V S; Kennedy P S; Presant C A; Smith J D; et al

CS Los Angeles Oncologic Institute, Los Angeles, California, USA; St. Vincent

Medical Center, Los Angeles, California, USA

SO Western Journal of Medicine (Sep 1, 1989), Vol. 151, pp. 283-288

DT (Clinical study)

RE Cancer Chemotherapy (Summary): Alert no. 1, 1990

FS Summary

LA English

WC 392

TX Purpose:

Intralymphatic inoculation of **autologous**, irradiated **tumour** cells has increased immunological responses in **breast** and renal **cancer** and has raised numbers of circulating CD4+ lymphocytes. In this study cyclophosphamide pretreatment was investigated as a possible inhibitor of suppressor cell activity, while the effects of incubation of cells with cholesteryl hemisuccinate, with the intent of increasing **tumour** cell-surface immunogenicity by reducing membrane-lipid microviscosity was also examined. In this

phase

I-II study, the efficacy of intralymphatic immunisation was studied in patients with advanced **cancer**, usually with pulmonary or intra-abdominal metastases.

Author comments:

Objective regressions were found in 7 of 32 patients, 5 of whom had undergone previous chemotherapy. '. . . those very ill patients all had tumors for which effective or even palliative therapy is marginal . . .'. 'The role of cyclophosphamide and of pretreatment of the **tumor** cells with cholesteryl hemisuccinate is still unclear.' 'This process did not amplify the response rate in our study. The incidence of clinical responses is not meaningfully different among the three series: 3 of 13 (23%) in series 1, 2 of 7 (29%) in series 2, and 2 of 12 (17) in series 3.'

'Our experience suggests that the method of intralymphatic immunotherapy is reasonably safe and technically feasible.'

Study details:

Design: multicentre, open

Features: patients were treated with autologous, cryopreserved, irradiated

tumour cells which were injected directly into the lymphatic system via cannulation of a dorsal pedal lymphatic channel

Control: drug combination comparison

Subjects:

Type: patients

No: 32

Groups: 3

Age: 26-78 (median 48) years

Sex: 8 female & 24 male

Characteristics:

11 patients received no previous treatment

Concomitant medication:

IM triethylperazine, 10-15mg prior to cyclophosphamide dose

Drug table:

Drug	Dose	Route	Frequency	Duration
Autologous, irradiated tumour cells	10-15 x 10 sup(6) viable cells/ml	ILy	q2-4w	4-24 weeks
Cyclophosphamide (3 days prior to vaccine)	300 mg/m sup(2)	IV	q2-3w	4-24 weeks

Results table:

	Group 1 (n=13)	Group 2 (n=7)	Group 3 (n=12)
Complete remission	1 (lung)	1 (melanoma)	0
Partial remission	0	0	1 (melanoma)
Mixed response	2 (melanoma, colon)	1 (colon)	1 (sarcoma)
Stable disease	2 (colon)	1 (melanoma)	0
Time to progression (weeks)	4-32	4-68	4-25
Survival (weeks)	6-300	10-169	6-59
Overall median survival =	36 weeks		

SIDE Side effects table:

Side effects (patients)

Wound infection	3	-	-
Difficult cannulation	3	-	-

PNO 32

GNO 3

CT **Cancer**, treatment

L4 ANSWER 23 OF 24 CANCERLIT

AN 79805339 CANCERLIT

DN 79805339

TI INDUCTION OF CUTANEOUS DHR TO IRRADIATED **AUTOLOGOUS TUMOR CELLS IN INOPERABLE BREAST CANCER.**

AU Adler A; Stein J A; Czernobilsky B

SO Non-serial, (1977). Prevention and Detection of Cancer. Part I. Prevention. Vol. 1. Etiology. Proceedings of the Third International Symposium on Detection and Prevention of Cancer held 26 April - 1 May

1976 New York NY. Nieburgs HE, ed. New York, Marcel Dekker, Inc., 1193 pp., 1977.

DT Book; (MONOGRAPH)

FS CATH

LA English

EM 197910

AB Autologous irradiated **tumor cell vaccine** (I-TCV) was prepared from biopsy specimens of breast **cancer** from 1 man and 22 women. Six patients had distant metastases; 17 had locally advanced, inoperable tumors and no evidence of metastases. Six areas near draining lymph nodes were injected id with a mixture of I-TCV (5 x 10(5) cells)

and individualized doses of BCG. Unmixed I-TCV (10(7) cells) was injected

into the center of the rosette. Immunotherapy (ImT) was given 1x/wk. Strong delayed cutaneous hypersensitivity (DCH) reaction to I-TCV, identical to DCH reactions to I-TCV/BCG, were elicited in all 17 inoperable patients after 2-6 wk of ImT. Max reactivity was seen after 6-9 wk. A lesser

degree

of DCH developed in 2/6 patients with metastases. Follow-up times were
2-8 mo, with booster doses (1x/mo) of I-TCV. Two patients needed additional
boosters of I-TCV/BCG to maintain their DCH reactions to I-TCV.
Suspensions of normal irradiated autologous skin or WBC elicited little

or

no DCH. DCH reactions to nonirradiated autologous **tumor** cells
and I-TCV were identical in 6/6 patients tested. Two inoperable patients
showed a flare-up reaction after 4 wk of ImT, simultaneously with the
first signs of DCH. These reactions proceeded to central necrosis. One of
these patients was given radiotherapy (3,000 rads) before the fifth and
last dose of I-TCV/BCG. The bulk of the **tumor** regressed. During
the last 4 mo of the 8-mo observation period, the patient had a recurrent
syndrome (every 3-4 wk) of fever, chills, malaise, and inflammatory
necrosis of microscopic **tumor** foci around, but not within, the
grossly visible **tumor**. ImT may favorably alter the host-
tumor balance only if the **tumor** is of microscopic size,
which would explain the absence of clinical **tumor** regression in
these patients with large tumors. ImT may be more effective against
micrometastases in postmastectomy patients. (5 Refs)